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MANAGEMENT OF A ONE MONTH OLD JAUNDICED INFANT

(Discussion of a Case Due to Inspissated Bile Which Cleared Spontaneously)

Case Report No. 112

Robert O. Warthen, M.D. L. W. 47–11963

L. W., a one month old colored male, was admitted to the Children's Hospital on November 14, 1947 because of jaundice of approximately 10 days duration. He was born in a nearby hospital on October 9, 1947 and had been well since birth with the exception of failing to gain weight. He was the product of a full term uncomplicated pregnancy and was delivered spontaneously after an 8 hour labor without ensuing neonatal complications. At birth he weighed 7 lbs. 7 oz. and although breast fed every three hours he weighed only 7 lbs. 2 oz. at the time of admission to the hospital. He had been receiving cod liver oil and orange juice daily since one month of age.

The history revealed no evidence of any illness since birth with the exception of the "yellowish tint to the skin and the eyes" which was first noticed 10 days prior to admission. The urine was noted to be "dark yellow" for 8 days preceding admission and since birth his stools had been "yellowish" although never pale or chalky. There had been no episodes of fever, vomiting, diarrhea, symptoms referable to the central nervous system, or constipation. The patient was the mother's first infant and there was no history of a previous pregnancy, abortion or transfusion. Neither the mother nor father had been treated for lues and the mother's Wasserman was reported negative $2\frac{1}{2}$ months prior to delivery of the patient. The mother was Rh positive.

The family history revealed no familial diseases or chronic illnesses with the exception of an attack of "infectious hepatitis" in the mother's 10 year old sister occurring one year previously and lasting approximately one month.

Physical examination on admission revealed a poorly developed, malnourished colored male with a greenish-yellow tint to his sclereae and an icteroid tint to his palate and mucous membranes. The pulse was 140 per minute, respirations 16 per minute and rectal temperature 98.6°. The remainder of the physical examination was essentially negative with the exception of a sinus tachycardia of 140 per minute, a liver palpable one and one-half finger breadths below the right costal margin and a moderate degree of phimosis. There was very little subcutaneous fat present. The stool was soft in consistency and yellow-white in color.

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On admission the infant was placed on a formula of Olac, cod liver oil and orange juice. The Van den Bergh at that time was immediate and direct and quantitatively revealed 2.9 mgm. % of bilirubin. The blood standard Kahn and Mazzini were negative and no urobilinogen was found in the stool or urine when tested by Ehrlich's method. The infant was Rh positive. Fachet's test revealed the presence of bilirubin in the urine.

During the subsequent 10 hospital days the Van den Bergh remained immediate and direct on two occasions and quantitatively revealed 3.4 and 3.5 mgm. % of bilirubin. Daily 24 hour urine specimens were negative for urobilinogen and positive for bilirubin and the stools became weakly positive for urobilinogen.

On the 18th hospital day a 24 hour stool specimen was positive for urobilinogen to the extent of 4 Ehrlich units or one milligram (normal for this age group being approximately 30 to 80 Enrlich Units or 8 to 20 mgm.). On the 20th hospital day, when surgery was contemplated, the stools assumed a greenish color and a delayed Van den Bergh with 1.8 mgm. % bilirubin was obtained. Because of this, surgery no longer seemed indicated and further diagnostic tests were deemed advisable. A repeat Kahn and Mazzini were negative. On the following day (21st hospital day) a 24 hour stool specimen revealed 18 Ehrlich units (4.5 mgm.) of urobilinogen.

The icteric tint of the sclerae persisted until the 36th hospital day and from the 20th to the 30th days the following tests were performed: Urine and urobilinogen, negative; tyrosine crystals in the urine, absent; cephalin flocculation, one plus at the end of 24 and 48 hours; blood cholesterol, 135 mgm. %; prothrombin time, 100% of normalcy; urine bilirubin, positive; coagulation time, 3 minutes; bleeding time, 1 minute; sickling smear preparation, negative after 48 hours.

On the 47th hospital day the Van den Bergh was immediate and direct with 0.9 mgm. % of blood bilirubin and the icterus index was 13.5. The 24 hour stool urobilinogen was 31.2 Ehrlich units (8 mgm.) and urine urobilinogen and bilirubin were absent.

X-rays of the long bones and chest on two occasions were reported as normal. A tuberculin test (O.T. 1/1000) was negative.

The infant was essentially afebrile (temperature varying from 97–100°) during the entire hospital stay with the exception of the 26th hospital day when he developed a fever of 101° associated with pharyngitis and mild diarrhea. The pharyngitis was controlled readily by penicillin and the diarrhea by powdered protein milk, clyses and symptomatic therapy. During this bout of diarrhea his red-cell count dropped to 2.5 million and his hemoglobin to 8.5 gms. and a transfusion of 75 cc. of whole blood was administered. With the exception of this episode the hemoglobin ranged

from 9 to 12.5 gms. and the red-cell count from 3.6 to 4.7 million. The white blood cell counts and differentials were all within normal limits. Routine urinalyses were reported as negative.

The infant gained weight steadily from the time of admission with the exception of a loss of two ounces during the diarrhea episode, and when discharged on the 54th hospital day he weighed 10 lbs.

His stools were of a pale yellow color until the 20th hospital day when they first became pale green-brown and remained so until discharge.

At the time of discharge he was receiving evaporated milk, dextrimaltose, cereal, orange juice and oleum percomorphum daily, and physically revealed no positive findings with the exception of a liver palpable one finger breadth below the right costal margin.

In a follow-up study 23 days after discharge from the hospital he weighed 11 pounds 14 ounces and physically was completely normal. At that time his Van den Bergh was indirect with 0.3 mgm. % of bilirubin, his urine revealed no urobilinogen or bilirubin, and a 24 hour stool showed 26 Ehrlich units (7 mgm.) of urobilinogen.

DISCUSSION

Upon being confronted with a case of jaundice at approximately one month of age the important decision rests as to whether a surgical problem is present. To that end one must first determine whether the jaundice is of a regurgitant or retentive nature.

Retention jaundice is due to liver damage or hemolysis of red blood cells and is characterized by greenish colored stools, a delayed indirect Van den Bergh reaction (due to a poorly functioning liver being unable to excrete bilirubin from the circulating blood), increased urobilinogen in the stool and urine and an absence of bilirubin in the urine. The high blood bilirubin is protein bound because increased bile salts in the blood stream due to obstruction below the liver are necessary for the splitting of protein from bilirubin and only protein free bilirubin can be excreted in the urine. Regurgitant jaundice is due to an obstructive lesion of the common bile duct or biliary tree and is characterized by yellowish to clay colored stools, an immediate direct Van den Bergh reaction, a decrease to absence of urobilinogen in the stool, an absence of urobilinogen in the urine and an increase in urinary bile salts and bilirubin. Due to the regurgitated bile salts, the protein is split from the blood bilirubin and the protein-free bilirubin is excreted in the urine.

The case here presented falls into the latter grouping and the discussion that follows will be directed along those lines. Inasmuch as the regurgitant nature of the jaundice is established the question of surgery now presents itself. At this point all laboratory procedures must be directed

towards that end and a minimum of such procedures is extremely important in order that the patient will be a good operative risk.

The most frequent causes of an obstructive jaundice at this age are congenital obstruction of the biliary tree and inspissated bile in the common bile duct; the treatment of choice in both of these conditions being surgery. Less frequent causes to be considered are infectious hepatitis, choledochal cyst, cholelithiasis, congenital syphilis, enlarged extrinsic lymph nodes and parasites.

The case presented is probably one of inspissated bile or mucus in the biliary passages which cleared spontaneously before surgery or duodenal drainage with magnesium sulfate was performed. The case will be discussed as one of inspissated bile or mucus; however, congenital atresia of the bile ducts will be discussed along with it inasmuch as the two conditions may be clinically indistinguishable and are often confused.

In both, the jaundice usually appears during the first few weeks of life (rarely at birth) and is often intense in character, the icterus index usually varying from 50 to 325 with an average around 100. The jaundice rarely appears initially after the third month of life and in most cases is present before the second month. In a few rare cases the intensity of the jaundice varies and when this occurs the diagnosis is almost certainly that of inspissated bile or mucus in that the intensity of the jaundice is continuous in cases of atresia.

The blood Van den Bergh is always immediate and direct with a blood bilirubin level of from approximately 2.0 to 50 mgm. %.

The stools are clay colored or white from birth with the exception of an occasional case with faint yellow stools due to the excretion into the intestinal tract of biliary pigments. If, however, an occasional faint green stool is noted one can more readily suspect inspissated bile or mucus due to the possible intermittency of that type of obstruction.

Urobilinogen is always absent in the stool of a case of atresia or of inspissated bile; however, in the latter case a small trace of urobilinogen may be rarely present when intermittency of the obstruction is the rule. In the case here presented urobilinogen began appearing in the stool in small quantities when the obstruction spontaneously relieved itself.

The urine is usually highly colored with biliary pigments and salts, and tests for urinary bilirubin are positive while urobilinogen is absent.

In both conditions there is usually a poor absorption of fats due to the absence of bile in the intestinal tract. Although in most instances the nutritional status is normal to excellent, such was not the case in this patient. It is believed that malnutrition and dehydration predispose to inspissated bile or mucus and these may have been the etiological factors in this case.

Bleeding tendencies are not the rule; however, in late unoperated cases over 5 to 6 months of age they may occur due to liver damage. The blood clotting time may be increased to 6 to 7 minutes. The liver is always enlarged, some ascitic fluid is usually present and the spleen may or may not be enlarged.

From the above discussion it now becomes evident that the only possible means (other than surgical exploration) to differentiate an obstruction due to inspissated bile or mucus from one due to congenital biliary duct atresia is by obtaining a history or clinical finding suggesting an intermittency of the obstruction.

According to Ladd and Gross the treatment of choice in either of these conditions is surgery, moreover it is considered extremely desirable to recognize the conditions early so that surgical relief can be instituted. In the case of biliary atresia an anastomosis of bile duct or gall bladder remnants with the gastrointestinal tract is performed if possible and in the case of inspissated bile or mucus one is often able to dislodge the plug by either manipulating the extrabiliary system or irrigating the ducts via the gall bladder. Ladd and Gross successfully treated a case of inspissated bile obstruction by fluoroscopically placing a tube into the duodenum and injecting 25% magnesium sulfate into the duodenum via the tube. This cholagogue apparently stimulated the flow of bile sufficiently to dislodge the material obstructing the common bile duct and subsequent to this, bile appeared in the stool, the jaundice cleared and there was no recurrence of symptoms. This method has not proved so successful in other cases and for that reason has been generally discarded.

The case here presented is one that cleared spontaneously while surgery was being contemplated, for on the 30th day after the jaundice was first noticed the stools suddenly became greenish-brown in color, the Van den Bergh became delayed with a bilirubin blood level of 1.8 mgm. % and the urobilinogen appeared in the stool in increasing significant amounts. As was mentioned in the protocol above, all of the findings gradually returned to normal.

Along these lines, it is believed that surgery should be delayed until the infant is at least 4 to 6 weeks of age if there are any findings suggestive of inspissated bile or mucus. This belief is based on recent reports in the literature which indicate that an obstructive biliary cirrhosis is not as important as previously believed in that prior to 4 to 6 weeks of age very little liver damage is incurred and this small amount of damage is readily repaired.

The average cases of bile duct atresia and inspissated bile or mucus if not operated or otherwise relieved will run a gradual downhill course and die between 5 and 12 months of age.

After the problem of surgery has been eliminated one may then feel safe to perform further laboratory examinations to establish a definite diagnosis. These tests will be considered later in the discussion.

Infectious hepatitis due to a virus is next considered as a possible cause of obstructive jaundice. This condition differs from atresia of the bile ducts or inspissated bile in that it is practically non-existent below the age of two years and extremely rare under two months of age due to the incubation period being over thirty days. It is presumably possible that the virus could be transmitted from the mother to the infant in utero; however, no such cases have been reported. This was considered because there was a family history of infectious hepatitis less than a year prior to birth. Infectious hepatitis is usually ushered in by a fever and vomiting, the fever receding after several days at which time the jaundice first appears. At the onset of the jaundice there is generally an increase in urinary urobilinogen (due to liver damage); however, in most cases there follows a complete obstruction to bile in the liver resulting in an absence of urinary and fecal urobilinogen. Later when the bile plugs are cleared from the liver there appears a normal amount of fecal urobilinogen and an increase in urinary urobilinogen, the latter being due to the inability of the liver to remove the urobilinogen from the blood (i.e. the urobilinogen that is absorbed into the blood stream from the intestinal tract) and again excrete it into the intestinal tract. The leukocyte count is usually elevated.

Additional liver function tests considered to be of value as evidence of liver damage and from a differential diagnostic point of view (after surgery has been eliminated) are listed below with the usual findings present in a case of infectious hepatitis.

Prothrombin time increased over 30 seconds; cephalin flocculation, 3–4 plus; cholesterol, normal to reduced; cholesterol esters, reduced to absent; tyrosine crystals, present in urine; Van den Bergh, immediate direct at first, changing to biphasic later, quantitatively 1.5 to 30 mgm. of bilirubin; thymol turbidity, 2.5 to 35 units.

Other toxic agents, i.e. chemical and vegetable, may produce a picture not unlike that of infectious hepatitis.

Another rare condition producing jaundice in an infant is idiopathic dilatation of the common bile duct (choledochal cyst); however, 95% of these cases are not discovered before 6 months of age. Here the diagnosis is based on an abdominal tumor plus all clinical signs and symptoms of obstruction of the bile duct.

Acute hemolytic anemias with jaundice are ruled out on the basis of finding an obstructive rather than a retentive type of jaundice. The latter are characterized by recurrent bouts of mild jaundice (due to a temporary saturation of the blood stream with liberated protein-bound

bilirubin), and increased fragility of the red blood cells, increased reticulocytes, increased urobilinogen in the urine and feces, absence of bilirubin in the urine, anemia during a crisis and an enlarged spleen. Rarely cholelithiasis will develop as a result of the excessive excretion of biliary pigment, the pigment forming a nidus for gallstones. In an unusual case such as this, abdominal pain, vomiting and a jaundice of the intermittent obstructive type are superimposed on the picture of hemolytic anemia described above.

Bacteremia may also produce a hemolytic type of jaundice (so-called "infectious jaundice"); however, these cases are generally readily detectable by associated signs of sepsis such as leukocytosis, fever and progressive anemia. The stool and urine are negative, the coagulation time is increased and the blood culture may be positive.

Congenital syphilis is ruled out in view of the absence of a maternal history of lues, positive serological tests in the mother and infant, positive long bone x-rays and demonstrable clinical stimata of syphilis.

Enlarged lymph nodes pressing on the common bile duct may also cause an obstructive type of jaundice. Parasites are also rare causes of obstruction.

Neoplasms of the liver such as carcinoma, hemangioma, hamartoma and adenoma are rare causes and are suspected on palpation of an upper abdominal mass.

Erthroblastosis foetalis must also be considered; however, in this instance it was a remote possibility because the mother and infant were Rh positive, this was the mother's first pregnancy and the mother had never received a blood transufsion.

Physiological jaundice rarely persists beyond two weeks of age and for that reason is readily ruled out.

The following is a suggested list of laboratory tests for diagnosis of a case of jaundice about one month of age:

1. Van den Bergh, quantitative and qualitative (and Icterus Index)
2. Urobilinogen in stool and urine, quantitative and qualitative.
3. Bilirubin in urine.
4. Serologic tests for syphilis—mother and infant
5. X-rays of the long bones.
6. Rh blood status of mother and infant.
7. Complete blood count.

After the problem of surgery has been eliminated the following laboratory examinations may be of additional value.

1. Erythrocyte fragility test.
2. Blood cholesterol and cholesterol esters.
3. Cephalin flocculation.
4. Prothrombin time.

5. Clotting time.
6. Tyrosine crystals in the urine.
7. Thymol turbidity.
8. Reticulocyte count.
9. Coagulation time.
10. Blood culture.
11. Bleeding time.

SUMMARY

1. Presented is a case of bile duct obstruction probably due to inspissated bile or mucus which cleared spontaneously without dudoenal drainage or surgical assistance.

2. The diagnosis, treatment and differential diagnosis are briefly discussed.

3. Important laboratory tests essential for diagnosis of a similar case are listed.

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SPECIAL REPORT

A REVIEW OF TWENTY-NINE CASES OF TETANUS AT CHILDREN'S HOSPITAL, 1927 TO 1946

Charles Green, M.D.

During the years 1927 to 1946 inclusive there have been twenty-nine patients with tetanus admitted to the wards of the Children's Hospital, Washington, D. C. It is the purpose of this report to present some observations on this disease with special reference to methods of treatment.

Incidence: Sixteen of the twenty-nine cases occurred in children from five to six years of age. Eight of the cases were ten to eleven years of age. As to sex, twenty-two cases were male and seven were females while seventeen were white and twelve were colored.

Portal of Entry and Incubation Period: It is interesting to note that in seven of the cases no portal of entry could be found after repeated examinations. In the remaining twenty-two cases various types of injuries were found. There were eight with lacerations, seven with puncture wounds and five with a splinter in one of the extremities and one with an infection of the umbilical stump.

In this series, the average incubation period of those who died was 6.2 days while the incubation period for those who recovered was 10.3 days. It has long been recognized that the incubation period has been an important factor in determining the prognosis, the short incubation period providing a poor prognosis and the long incubation period giving a more favorable outcome. Two exceptions were noted to this general rule. One patient (Case 23) had an incubation period of nine days and after vigorous therapy was apparently improving when he suddenly developed severe respiratory distress. The temperature, pulse and respiration rose rapidly to 107°, 140 and 60 per minute respectively. The patient had evidently aspirated some fluid which had been given by tube feeding. Roentgen examination of the chest was reported to show a massive collapse of the entire right lung. Breath sounds were accentuated in the left chest and dullness was elicited in the right. The patient was bronchoscoped and thick mucus was aspirated from the right upper main bronchus. However this did not seem to relieve the respiratory embarassment and death occurred a few hours later, eighteen days after the injury and 9 days after the onset of symptoms. The other exception was that of a six year old child (Case 7) with an incubation period of four days who survived. This child gave a definite history of receiving tetanus toxoid at the age of three years which probably modified the course of the disease.

The average duration of hospital stay for those who died was 4.5 days while the average stay for those who recovered was 26.6 days.

Presenting Symptoms: The most common presenting symptom was trismus or stiff jaw which was present in 79% of the cases. The next most common complaint was stiffness of the neck, back and legs. This was noted in 75% of the cases. Eight of the cases had difficulty in swallowing. Six cases were in opisthotonus on admission and in one case the presenting symptom was a convulsive seizure.

Laboratory: Laboratory work on these patients consisted of blood counts, urinalyses and in some cases lumbar puncture and cultures of the material from the wounds. The average hemoblobin in these cases was 11 grams.

TABLE 1

Distribution of cases and fatalities according to age groups

AGE	NO. OF CASES	DEATHS
years		
0– 4	3	2
4– 8	17	7
8–12	9	3

TABLE 2

Racial and sex distribution

	NO. OF CASES	MALE	FEMALE
White	17	11	6
Colored	12	11	1

The leucocyte counts ranged from 6,000 to 14,000 cells per cmm. Urinalyses on all patients were essentially negative. Lumbar punctures were performed in nine patients and were normal in every case.

Cultures of material from the wounds were made in six of the twenty-nine cases. In one case a hemolytic streptococcus and a hemolytic staphylococcus was isolated. In two cases no anaerobic organisms could be cultured. One case revealed an anaerobic hemolytic streptococcus and a Gram positive bacillus with subterminal spores. In two cases a positive culture of Clostridium Tetani was isolated. In one case the tissue excised from the wound was sent to the U. S. Public Health Laboratory which reported a positive culture of Clostridium Tetani from the specimen.

Treatment: The patients were kept in a quiet, darkened room and external stimuli eliminated as much as possible. In most instances nurses

were in constant attendance. Aspirators were available in each room and oxygen was used freely in those patients presenting respiratory distress. Debridement of the wound was performed in 14 of the 15 cases having a demonstrable wound. Following debridement, 10,000 to 20,000 units of

TABLE 3

Correlation between incubation period and outcome

CASE NO.	INCUBATION PERIOD	OUTCOME
	days	
1	12	R
2	11	R
3	5	D
4	7	D
5	10	R
6	14	R
7	4	R
8	Unknown	R
9	7	D
10	Unknown	D
11	6	D
12	7	R
13	12	R
14	19	R
15	7	D
16	5	D
17	7	D
18	Unknown	R
19	18	R
20	10	R
21	Unknown	D
22	6	D
23	9	D
24	5	R
25	7	R
26	Unknown	R
27	3	D
28	Unknown	R
29	6	D

antitoxin was infiltrated in the area around the wound. Local treatment consisted of irrigations with hydrogen peroxide, and dressings with boric acid or sulfonamides. In three cases, 10,000 units of penicillin was also infiltrated into the area about the wound. In one case in which there was a wound of the upper lip no debridement or infiltration with antitoxin was performed.

Tetanus antitoxin was administered to all patients shortly after admission. The initial dosage varied from 20,000 to 100,000 units depending upon the severity of the presenting symptoms and signs. The average total dosage for those that died was 100,000 and the average for those that recovered was 176,000. Vinnard[2] in his series concluded that the maximum effective initial dose of antiserum is about 80,000 to 100,000 units. Pratt[1] states that he found no evidence that 80,000 units were more effective than 30,000. His observation is in accord with the immunologic data of Spaeth[4]. However, Vener and Bower[5] reported 100 cases of tetanus with low gross mortality rate of 29% and advised a minimum of 200,000 units of antitoxin.

TABLE 4

Incidence of presenting symptons

SYMPTOM	INCIDENCE
	%
Trismus	79.3
Stiff neck, legs, back	75.8
Dysphagia	27.6
Opisthotnus	20.6
Convulsion	3.4

TABLE 5

Route of administration of tetanus antitoxin

	NO. OF CASES	DEATHS
Intravenously only	2	2
Intramuscularly only	5	1
Intravenously plus intramuscularly	12	4
Intrathecally plus intramuscularly and intravenously	10	5

Tetanus antitoxin was administered intrathecally to 10 of the 29 patients and 5 of these survived. However, the intrathecal route was subsequently abandoned and has not been used at Children's Hospital since 1932. The concensus of current opinion would indicate that the intraspinal route is often dangerous because of the excitement induced, the technical difficulties encountered in highly spastic patients and the possibility of introducing a sterile or septic meningitis. Pratt[1] indicts the intrathecal injection of tetanus antitoxin as chiefly responsible for the deaths of two patients in his series. Dietrich[6] felt that many patients with tetanus, some of them with mild infections, died much sooner after being treated with intrathecal injections of tetanus antitoxin than they could have from

untreated tetanus. The results of therapy according to the route of administration of tetanus antitoxin is summarized in Table 5 and illustrates the apparent superiority of administration of tetanus antitoxin intramuscularly and intravenously.

In this series, one case showed an immediate reaction to the intravenous injection of antitoxin. This reaction was manifested by a rapid pulse, circumoral pallor and respiratory distress. The reaction was controlled by injection of 0.5 cc. of adrenalin and the use of oxygen. One patient had a delayed serum reaction characterized by urticaria that developed one week after injection of tetanus antitoxin. Both of these patients survived.

Penicillin was used in 4 of the 29 cases, varying in total amounts from 750,000 units to 950,000 units. Three of the four patients died and it appears that the administration of penicillin had little effect on the course of the disease other than as a prophylaxis against secondary infection and pneumonia. These results are in agreement with those obtained by Altemeier[7] who concluded from a series of 16 cases of tetanus treated with

TABLE 6

1927 thru 1946

NO. OF CASES	RECOVERY	DEATHS	% MORTALITY
10	5	5	50
19	12	7	36.8
29	17	12	41.3

penicillin that the use of this drug failed to exert any beneficial effect in uncomplicated cases of tetanus.

Various types of sedation were employed in the treatment of the patients in this series. In general, paraldehyde, chloral hydrate, and sodium phenobarbital were used to control the convulsions. In some of the more recently treated cases, avertin was used and provided good sedation. This drug was given in doses of 60 to 80 milligrams per kilogram of body weight every 6 to 8 hours. In one case in this series curare was used but the total dosage was so small that it would be difficult to evaluate the amount of relaxation that was obtained.

There was a mortality rate of 41.3 per cent in this series (Table 6). This figure is comparable to numerous reports in the literature. A study of fifty six cases by Pratt[1] at the Children's Hospital in Boston revealed a gross mortality of 43 per cent. Vinnare[2] made a study of three hundred fifty-two cases at the Charity Hospital in New Orleans and reported a fatality rate of 45 per cent while Silverthorne[3] reviewed 70 cases admitted to the Children's Hospital of Toronto and found a mortality of 47 per cent.

The further reduction in the incidence of tetanus among children can be accomplished only by producing and constantly maintaining a high degree of active immunity against this infection. The recovery of one patient with an incubation period of 4 days but who had previously received toxoid suggests that active immunization may be a life-saving measure. The desirability of routinely immunizing all children against tetanus cannot be overemphasized.

SUMMARY AND CONCLUSIONS

1. Twenty-nine cases of tetanus are reviewed with a gross mortality of 41.3%.

2. The incubation period is the most important factor in determining the prognosis.

3. Adequate total dosage of tetanus antitoxin in this series ranged from 100,000 to 300,000 units. Our experience and that of others indicate that dosages under 100,000 are probably as adequate as larger doses.

4. Penicillin apparently does not alter the course of tetanus but may be used to combat secondary infectious processes such as pneumonia.

5. Avertin and various barbiturates are the drugs of choice for sedation.

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URETHRITIDES IN INFANTS AND CHILDREN

(Presentation of a Case of Streptococcic Urethritis)

Case Report No. 113

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Robert O. Warthen, M.D.

A. F. 47–9552

A. F., a 6 year 5 month old colored male, was seen in the Outpatient Department of the Children's Hospital on January 22, 1948 because of a purulent urethral discharge of one days duration. For two days prior to admission the mother had noted that the penis was sore to the touch and the child complained frequently of burning on urination. There was no history of sexual contact, uncleanliness, introduction of a foreign body into the urethra or trauma to the penis. There was no frequency, urgency or nocturia. The child had apparently been well with the exception of a chronic hypertrophic tonsilitis which was noted for the first time four days prior to the onset of the present illness when he was seen in the clinic for a general check-up.

He had had the usual childhood diseases including measles, whooping cough and chicken pox several years previously and had been subject to "several colds" a year since one year of age. Four months prior to the present illness he had been treated in the dispensary for minor abrasions of the left hand, left arm and left knee which healed readily.

He was born in a nearby hospital on August 29, 1941 after a full term pregnancy. The delivery was spontaneous and uncomplicated and there was no neonatal jaundice, anemia, cyanosis, petechia or convulsion. His physical and mental development followed along normal patterns and his dietary history was adequate.

There was no family history of lues, gonorrhea, tuberculosis or any other disease. No history of a direct or indirect venereal contact could be elicited.

Physical examination revealed a well developed, well nourished six and one half year old colored male not appearing acutely ill. The pulse was 80 per minute, respirations were 16 per minute and the temperature was 99.6°F. orally. All positive physical findings were limited to the head and neck and genitourinary system. The throat was midly inflammed as were the tonsils which were moderately hypertrophied and cryptic. One plus submaxillary and cervical glandular enlargement was present. The glands were firm and non-tender. The penis revealed the prepuce to be approximately $\frac{1}{4}$ retracted with the remaining $\frac{3}{4}$ bound tightly to the glans by adhesions. A yellowish thick creamy purulent discharge exuded from the

urethral meatus and upon milking the urethra this discharge flowed copiously. The urethral meatus was reddened and slightly edematous, the inflammation extending approximately 2 mm. onto the glans penis. There was no evidence of infection of the prepuce or glans adjacent to the prepuce. There was no abdominal or costovertebral angle tenderness. The inguinal lymph nodes were slightly enlarged. A smear of the purulent exudate revealed numerous gram positive cocci in long chains (later proven to be non-hemolytic streptococcus by culture). Another smear and culture taken at the same time were negative for gram negative intracellular diplococci. Upon finding the streptococcus, sulfadiazine therapy in a dose of one grain per pound per day (in divided doses given every 4 hours) was instituted and within sixteen hours of therapy, after having received $22\frac{1}{2}$ grains of sulfadiazine, the discharge from the urethra had ceased. Twenty-four hours after therapy was begun the preputial adhesions were separated with ease and no evidence of urethral inflammation was present. Urinalysis at that time was essentially negative and revealed a specific gravity of 1.030, an acid urine and a few amorphous crystals. At no time was a foreign body palpable in the urethra and rectal examination was essentially normal. The child was maintained on sulfadiazine for a five day period and at the end of that time a repeat urethral smear and culture as well as a urinalysis was reported as negative. There had been no recurrence of the urethral discharge at the time this paper was written.

DISCUSSION

When we are presented with a case similar to the one above, we may be confronted with many problems. However, we can best discuss such a case by covering the subject of urethritis briefly.

In such a discussion we must consider the types of which two are usually mentioned, gonorrheal and non-gonorrheal. The second group, to which this case belongs, has also been called simple and non-specific.

Within the non-gonorrheal group, of greater incidence in infants and children than the gonorrheal, we have many etiological factors which include uncleanliness, phimosis, balanoposthitis, highly acid urine, foreign bodies, masturbation or prostatitis. The organisms found upon smear and culture examinations may include staphylococci, streptococci (as in our case), B. coli, or diphtheroids as well as tubercle bacilli.

Gonorrheal infection has captured much of our literature on the urethritides because of its high incidence in adults and the nature of the infection. For that reason we will discuss the signs and symptoms of this type for comparison. This form of urethritis is due to the gram-negative diplococcus (gonococcus) and is very common. Many small children and even infants are frequently brought into the dispensary with complaints of a thick yellow or

yellowish green urethral discharge. Though a history is rarely elicited most infections must be from contact with fomites or by sexual or non-sexual contact with infected individuals. A careful history may reveal such contacts. Infection of the pre-adolescent urethra in the male probably always occurs from direct contact. Usually between the fourth and eighth days, by the time the infection has spread from the meatus to the deep anterior urethra, the invasion is manifested by urethral itching with burning on urination. Several days later the previously mentioned yellow creamy purulent discharge will appear and if the severe burning and itching has not sent the patient to the pediatrician, the discharge will. After two weeks an untreated infection may reach its height and if there is no urethral obstruction or other complication it may disappear spontaneously in six months. This is explained by the fact that the toxins of the gonococcus produce a tissue reaction of the urethra with which they come in contact, the purpose of this reaction being the elimination of the cause and the reparation of the damage that has resulted. Such spontaneous cures may be greatly prolonged if phimosis, congenital meatal or deep urethral stricture or other allied conditions exist. In non-gonorrheal urethritis we find as a rule that the same symptoms and signs, though milder, are present; however, they usually disappear with cleanliness.

The diagnosis of gonococcal urethritis is made by the demonstration of gonococci in the urethral discharge on culture. Smears are frequently unreliable and had best be regarded as only suggestive but not confirmatory of the diagnosis.

Tuberculous urethritis is very rare and would be found only in late advanced urogenital tuberculosis; however, again it is a question of finding the corresponding organism.

Foreign bodies in the urethra of infants and children usually occur without a history of introduction. Palpation of the urethra and x-ray examination for radio-opaque objects may be of value in locating the foreign body. In recent years the techniques of aero-urethrography and urethrography by means of contrast media have been developed and are of value in certain selected cases. A urinary stone, after having passed from the kidney and bladder, may become impacted in the urethra with a resultant urethritis and periurethritis and possibly infiltration of urine into the surrounding tissues.

The treatment of the urethritides is generally universal for all types. Prophylaxis requires cleanliness, circumcision in those cases of phimosis and meatotomy in congenital meatal obstruction. Adhesions and bands should be removed and again, of utmost importance, cleanliness should be stressed for cases of non-gonorrheal urethritis may have their origin with that failure. Many mothers have not been instructed as to the methods

and importance of maintaining cleanliness when leaving the hospital with a newborn. Foreign bodies must always be considered as mentioned above. These can be seen and removed when they occur if the mother cleans and examines the child frequently, otherwise time may pass and severe infections occur with more difficulties following.

In the treatment of gonorrheal and non-gonorrheal urethritis, cleanliness, rest and diet are important. Rest may limit the spread of the infection as well as combat the disease. Condiments and alcohol should be avoided. Forced fluids will increase urination thus cleansing the urethra.

With our present knowledge of chemotherapy and penicillin the specific infections may be readily treated; however, smears and cultures should always be performed. The choice of the sulfa drug lies between sulfathiazole and sulfadiazine, most authors preferring sulfathiazole. The drug should be continued for 5 consecutive days. In gonorrheal vaginitis the sulfa may be continued for 7 days. Usually within these periods, 5 to 7 days, there will be marked improvement in the discharge and the physical signs; however, if this is not true one must suspect sulfonamide fastness, prostatitis and other complications (which are rare in children). In this 10% sulfa fast group penicillin has proved of value and is oftentimes used without a previous trial of the sulfonamides. Soap and water have always been foes of the gonococcus. Urinary antiseptics are of very little value and may upset the gastrointestinal tract. As mentioned previously, fluids may cleanse the urethra and alkalinization may aid dysuria.

A dorsal slit or circumcision is indicated when balanoposthitis or phimosis is marked and meatotomy is of value when the urethral orifice is tight.

With the use of chemotherapy many of the older and reliable injection methods have been discarded and yet it might be well to discuss them for they are still valuable in chronic urethral gonorrhea as well as some of the chronic non-specific urethritides which have been treated adequately without success. Here we must inject a note of caution since some complications are caused by injudicious instrumentation or injections while active gonococci are present in the urethra; however, such injections may be of value in a chronic case when gonococci are no longer present. A mild silver proteinate (neosilvol or argyrol) 5 to 10%, or potassium permanganate 1:6000 to 1:8000 may be used. Neosilvol should be fresh and not over two weeks old when used. A blunt nose urethral syringe is used to instill one to three cubic centimeters of neosilvol or argyrol slowly into the urethra twice daily. This is retained there for three to five minutes. These instillations are continued until the urine is clear at which time they are reduced to one daily and can be discontinued in a week if there is no further evidence of infection. Urethral discharge which is non-gonorrheal

in type may be difficult to cure in a neurotic type child. The response to chemotherapy may not be too great where foreign bodies or disturbances with drainage of the urinary tract are present. In all cases treated with any of the methods recommended, the criterion of cure should be four consecutive negative cultures at two week intervals.

The complications of the two groups are identical, being less common and usually less severe in the non-venereal conditions. In 75% of the cases, posterior urethritis develops but the associated prostatitis is usually far less severe than in adults. Periurethritis or a periurethral abscess may occur in the acute stage. These and periurethral phlegmon are rare and are often a part of stricture pathology which is a late complication. Epididymitis occurs in about 5% of all cases of urethral gonorrhea in boys. Complicating gonorrheal proctitis occurs in 2 to 4% of females with specific vaginitis but is less common in gonorrheic males. Proctitis is treated by rectal irrigations of warm potassium permanganate solution (1:2000) twice daily. Gonorrheal septicemia with or without endocarditis is very rare and may be fatal.

In cases where treatment has been delayed or a foreign body not recognized, cystitis, pyelitis and other complications of the upper urinary tract may develop.

Another condition deserving of mention is the so-called "Reiter's disease". This syndrome consists of non-gonorrheal urethritis, conjunctivitis and arthritis and until recently has been almost completely ignored in the literature. It is more commonly seen in adults; however, a few cases in children have been reported. It is almost unheard of in the negro race.

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SUPPURATIVE PAROTITIS IN AN INFANT

Case Report No. 114

Frederic G. Burke, M.D. M. G. 48-301

M. G., a 3 week old infant, was admitted to the Children's Hospital on the 8th of January, 1948 with the chief complaint of "swelling in front of the left ear." The infant was the second child and had been born by spontaneous delivery without the use of instruments and her course in the hospital after delivery was uneventful. Twenty-four hours before admission the mother noticed a slight swelling in front of the lower part of the left ear. The mother stated that it extended upwards to the level of the mid-portion of the auricle and anteriorly about 2 inches in front of the external auditory canal and downwards to the angle of the mandible. The infant had been somewhat restless but slept fairly well that evening and overnight the mass doubled in size. The mother and father and one sibling who was 15 months old were quite well and there was no history of contact with mumps. An interesting point was mentioned by the mother that she had had a similar swelling of her face shortly after she was born.

On physical examination this 3 week old infant did not appear to be in acute distress. The right side of the face was normal and there was an indurated, hard, apparently somewhat tender mass involving the left side of the face displacing the lower lobe of the ear laterally and posteriorly. The orifice of Stenson's duct was elevated and indurated and pressure upon the mass in the region of the parotid gland produced greenish-yellow pus from this opening. There did not seem to be a lack of secretions in the mouth itself and there was no sign of poor oral hygiene nor was there any evidence of dehydration. The baby was breast fed and upon admission to the hospital she was placed on a Similac formula.

X-ray examination of the involved region revealed no evidence of radio-opaque calculus or bone involvement. The blood count showed a leukocytosis of 20,000 cells but was otherwise essentially normal. Urinalysis showed 5 mgm. albumin with many white blood cells. Culture of the pus exuding from the left Stenson's duct revealed hemolytic Staphylococcus albus and non-hemolytic Streptococci.

The infant was given 30,000 units of penicillin intramuscularly every 3 hours for 4 days and sodium sulfadiazine grains 1½ per pound of body weight at divided 6 hour intervals. Her temperature upon admission was 100.6° and subsequently during her 5 days in the hospital became normal and remained within normal range. Thirty-six hours after therapy had been started the induration resolved, the mass became considerably smaller and

finally assumed normal size on the 4th day. Twenty-four hours after admission free pus was no longer noticed from the duct and in the second seventy-two hour period all local inflamation and induration subsided. Because of the rapid response to medical treatment the duct was not probed for evidence of a non-opaque calculus.

DISCUSSION

Suppurative parotitis is most commonly seen in adults following surgical operations particularly those of an abdominal nature. Parotitis other than mumps parotitis is very uncommon in infancy, only two such cases having been noted at the Children's Hospital in the past 5 years. It is interesting to speculate on how it is possible for this infant to have developed this infection since the conditions that have been noted in previously reported similar cases did not hold true in this instance. Trauma has been mentioned as a possible cause brought about by the use of instruments on the head at the time of delivery with resulting irritation by pressure. This child was delivered spontaneously. There was no evidence of gross infection of the mouth; the mucus membranes and tongue were well protected by an adequate amount of salivary secretions and there was no evidence of thrush or other type of stomatitis. There was no evidence of atresia of the glands secreting into the mouth nor was there any lack of normal secretion of the salivary juices. The lack of any external infection fairly well places the site of entrance at the orifice of the duct and probably represents a chance infection possibly from a contaminated nipple or breast.

The rapid response to medical treatment was quite gratifying since previously reported cases for the most part were treated surgically. Treatment was instituted on this baby within 24 hours after the mass first appeared and before she had become dehydrated and very ill. Thus the high mortality which is frequently mentioned with this condition did not come into play. After fluctuation has occurred surgical incision is usually indicated. In the one other case noted in this hospital three years ago an associated stenosis of the Stenson's duct was surgically corrected by incision and drainage of the duct and also resulted in rapid recovery with associated chemotherapy.

Of interest is the mother's history of a similar infection shortly after her own birth. The details were vague and it was not possible to confirm whether or not she actually had had parotitis but it may be speculated that this infant's parotid duct orifice might be congenitally narrow on an hereditary basis because of the rarity of this condition in normal infants. If such an anomaly exists in this baby, recurrence might be expected.

INGESTION OF FURNITURE POLISH

Case Report No. 115

Robert B. Sullivan, M.D. D. T. 47–2270

During the year 1947 there were sixty-six admissions to the Children's Hospital because of poisoning. Most of these children were not very sick and were admitted for observation in accordance with the policy of the hospital. About one half of these poisonings were due to the ingestion of volatile liquids of which kerosene was the most common. Furniture polish was the offending substance in the case presented here. This preparation was a mineral oil suspension in water which contained essential lemon oil and a kerosene-like volatile hydrocarbon.

On March 7, 1947 a fifteen month old colored female was admitted to the hospital because of rapid breathing and somnolence. Thirty hours before coming to the hospital she drank about one ounce or less of Old English furniture polish. Whether she coughed and aspirated some of it is not known. She was immediately made to drink milk but she did not vomit. There were no immediate ill effects but within three hours she became drowsy and during the night her respiratory rate was rapid. During the fifteen hours preceeding admission her respirations gradually became more rapid and she became slightly stuporous. She drank fluids readily and did not vomit. There was no pain or diarrhea or other complaint. Fever mounted throughout the day. Before this episode she had been a normal, healthy infant with an uneventful past history.

When admitted to the hospital, her temperature was 103.8° and the patient was drowsy but not stuporous. She was well developed and nourished and not dehydrated. Respirations were deep and the rate was about 60 per minute. There was no acetone odor or other aromatic scent to the breath. The pharynx seemed slightly reddened but there was no evidence of an upper respiratory infection. There was slight dulness over the right lung base posteriorly and a few fine rales were heard over the left lung base posteriorly. Except for a rapid rate the heart was normal. The abdomen, skin, extremities and neurological examinations were negative.

The red blood cell count was 3.7 million with 11 grams of hemoglobin. The white blood cell count was 8,700 with 55% polymorphonuclear forms, 4% band forms, and 41% lymphocytes. The urinary output was adequate, and the only abnormality found in the routine examination was a trace of acetone. An x-ray of the chest showed disseminated infiltration bilaterally.

During the first twenty-four hours her condition seemed quite good in

spite of rapid respirations (about 60–70 per minute) and fever (101° to 103°). Penicillin was given (40,000 units intramuscularly in the first dose followed by 25,000 units intramuscularly every three hours) in anticipation of secondary bacterial invasion of the chemical pneumonitis. Although

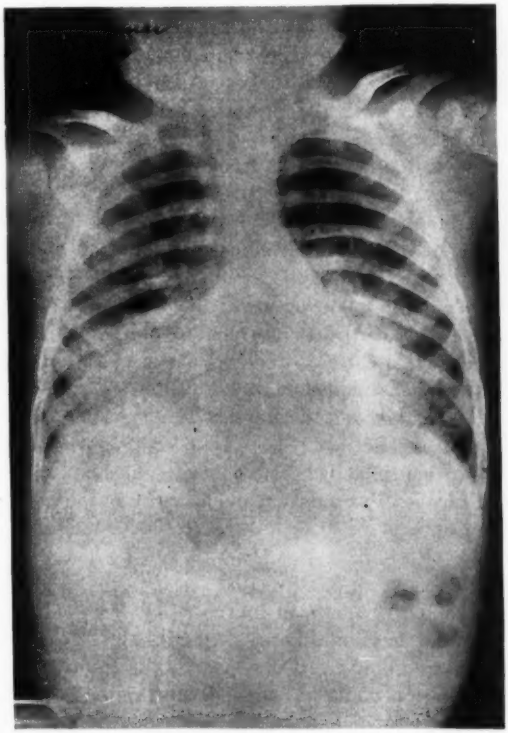

FIG. 1. D. T. Consolidation of the right base with marked bilateral interstitial pneumonitis due to ingestion of furniture polish. The lack of peripheral involvement would suggest the parenchymal damage occurred at the time of ingestion rather than an excretory effect.

the infant continued to take fluids by mouth freely a clysis of 150 cc. of 5% glucose in Hartman's solution was given and repeated eight hours later. During the second day of hospitalization her temperature rose to 105° and was sustained. The respiratory rate was irregular but was as high as 120 per minute much of the time. The carbon dioxide combining power was 38 volumes percent. She was given 200 cc. of $\frac{1}{6}$ molar lactate solution

intravenously and ten hours later 200 cc. of Hartman's solution subcutaneously. A second carbon dioxide combining power estimation done twenty-four hours after the first one was 53 volumes percent. Acidosis was considered to play no part in the hyperpnea. Antipyretic measures were employed vigorously. Water and alcohol sponge baths, cool water enemas and aspirin had little effect on the sustained fever during the first two days. Therefore these measures were abandoned and a cooled oxygen tent was used. In addition cold sheet packs were applied during the next three days. On the third day the fever became remittent and respirations which had been varying from 130 to 170 per minute began to decrease. During this time she had a mild diarrhea of four to six large green liquid stools, a transient albuminuria of 100 milligrams percent, and a mild anemia of 3.1 million red blood cells. All of these changes resolved spontaneously within one week. Another x-ray taken on the fourth day of hospitalization (see Fig. 1) showed considerable progression of the bilateral infiltration. During the following weeks the temperature was normal and the pulse and respiratory rates returned to normal in three days. An x-ray of the chest taken on the 13th day of the disease showed the lesion to be resolving. Dulness, diminished breath sound transmission and moderately coarse rales could be demonstrated bilaterally but clinically the infant seemed quite well and active. She was discharged on the 16th day of hospitalization to continue her convalescence at home. She recovered completely. A physical examination and an x-ray of the chest nine months later showed her to be in excellent health.

DISCUSSION

In the July issue of the American Journal of Diseases of Children, Steiner presented 35 cases of accidental kerosene poisoning. He divided the cases into three groups according to their severity. In group I there was acute toxicity and depression of the central nervous system with minimal pulmonary changes and rapid uneventful recovery. In group II there was severe pneumonia, hyperpyrexia and prolonged recovery. In group III there was severe pneumonia with evidence of degenerative changes in the myocardium, liver, kidney, and gastrointestinal tract. It is hoped that familiarity with these syndromes will influence the handling of future cases of kerosene and similar poisonings admitted to the hospital. There is experimental evidence that the pulmonary changes in such cases are caused by both aspiration of the toxic fumes and fluid and by absorption of the fluid into the blood stream. The most recent opinion is that gastric lavage with copious amounts of a weak sodium bicarbonate solution is the best immediate treatment. Caffeine has been used to combat the central depression of which drowsiness is the characteristic sign. These may be

rapid, feeble pulse, shallow respirations, cyanosis, muscular twitchings and stupor or coma. Pneumonia can be demonstrated by x-ray in cases where the ingested amount was extremely small and where clinical signs and symptoms are absent. Marked pulmonary changes may necessitate the use of oxygen for hyperpnea or cyanosis. Penicillin is employed to forestall secondary bacterial invasion. Hyperpyrexia may be extreme and difficult to control. Cold packs and adequate hydration are more effective than antipyretics. Myocardial damage may be demonstrated by the electrocardiogram and in one case auricular fibrillation and flutter occurred. Congestive heart failure as evidenced by dilation of the heart, enlargement of the liver and elevation of the venous pressure may occur. This should be treated by digitalization. Kidney damage may be demonstrated by albumin, casts and white blood cells. Adequate oral or parenteral fluid intake will probably minimize this damage. Gastro-intestinal mucosal changes are shown by evidence of blood in vomitus and by bloody diarrheal stools. Prompt gavage soon after ingestion is the only effective preventive therapy. Children whose poisonings are severe enough to be classified in group III are extremely ill for five to ten days and seem to be in imminent danger of dying. However, the mortality rate is quite low and all of the systematic changes mentioned above are reversible and there are apparently no residuals.

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